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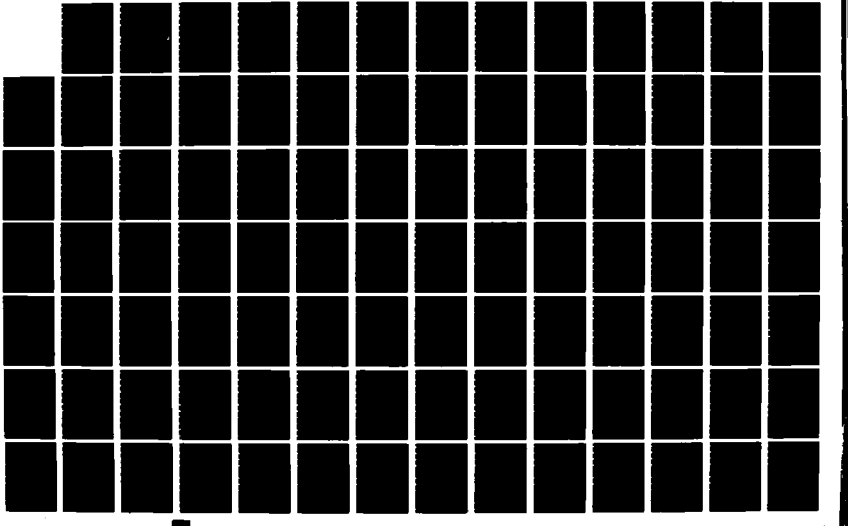

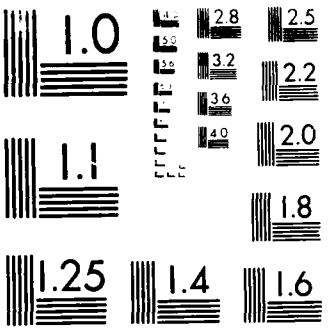
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Abstract

The Effect of Cigarette Smoking on
Gingival Crevicular Fluid Flow

by

Laurence Paul Crigger
LtCol, United States Air Force

1986

88 pages

M.S.D.
Indiana University School of Dentistry

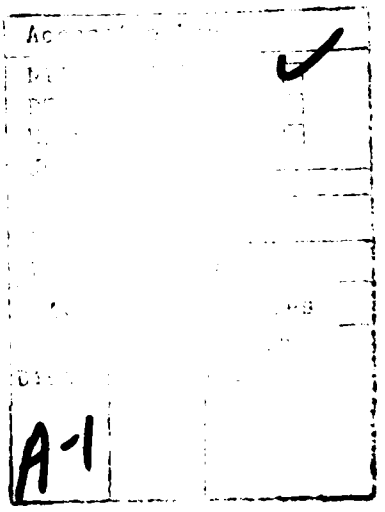
Gingival crevicular fluid (GCF) flow rates were measured with a Periotron 6000 in 60 smokers and 49 nonsmokers. In addition, carbon monoxide (CO) concentration of expired air was measured, and plaque and gingivitis indices were recorded for all subjects. All subjects completed a medical history and a smoker's questionnaire. Smokers also completed the Fagerstrom Tolerance Questionnaire.

Differences in GCF flow between smokers and nonsmokers were not statistically different. Smokers had a higher concentration of CO in expired air, more plaque accumulation, and a higher gingivitis score than nonsmokers. The differences in all three parameters were highly significant.

GCF was positively correlated with gingivitis scores, but plaque scores showed a stronger correlation in both groups. GCF showed no correlation with either carbon monoxide levels or the number of cigarettes smoked per day.

There were strong positive correlations between Fagerstrom scores and daily tobacco consumption, as well as between carbon monoxide levels and both daily consumption and lifetime consumption as measured by pack years. Still positive, but slightly weaker correlations were found between GCF flow and plaque accumulation, between carbon monoxide levels and Fagerstrom scores, between carbon monoxide levels and years smoked, and between Fagerstrom scores and both pack years and years smoked.

Negative correlations, albeit weak ones, were found between carbon monoxide and both plaque and gingivitis prevalence.



THE EFFECT OF CIGARETTE SMOKING ON GINGIVAL CREVICULAR FLUID FLOW

by

Laurence Paul Crigger

Submitted to the Faculty of the Graduate School in partial fulfillment of the requirements for the degree of Master of Science in Dentistry, Indiana University School of Dentistry, 1986.

Thesis accepted by the faculty of the Department of Preventive Dentistry, Indiana University School of Dentistry, in partial fulfillment of the requirements for the degree Master of Science in Dentistry.

Melvin R. Lund

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Chairman of the Committee

Date June 17, 1986

Acknowledgments

I would like to express my gratitude and appreciation to the one person around whom everything I do centers and against whom I measure all success - my wife Sally. For 19 years we have shared a life together that only improves with time. May it be everlasting. Thank you for being my wife, my lover, my best friend, and for just being.

I must also recognize the contributions of three men who have influenced my Air Force career in the most positive of ways. To Colonel Dale Granger, who took a once rebellious young officer and pointed him in the right direction; to Colonel Paul Park, who allowed that officer to stretch and grow and demonstrated that nice guys can finish first; and to Colonel John Young, the most gentlemanly of scholars who encouraged that same officer to strive for loftier heights, I thank all of you for the time and effort expended on my behalf. You may not have known it, but you've left your mark.

I wish to thank Major Robert Bousquet, my co-investigator in this study, and his wife Sandy, who made my stay at Chanute Air Force Base an enjoyable one.

Also I owe a debt of thanks to Dr. Arden Christen, whose unfailing confidence in me has meant a great deal. I know this will be only the beginning of a long and fruitful association.

Finally, I would like to thank the members of my committee, Dr. Lund, Dr. McDonald, Dr. Olson, and Professor Barton, for their support and assistance.

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Introduction

Since the original Surgeon General's report was issued over 20 years ago, cigarette smoking has been linked to a wide variety of systemic diseases - lung cancer, heart disease, and emphysema to name but a few. In addition to the harmful sequelae that may befall a smoker, there is growing evidence to indicate that the use of tobacco products may also be a health risk for persons who live or work with smokers, but who themselves do not indulge directly in this practice. The danger may even extend to the unborn child of a pregnant woman. Indeed the smoking habit poses considerable risks to smokers and nonsmokers alike.

When one ponders the adverse consequences associated with smoking, risks to the oral cavity are often overlooked. Even more remotely considered are the deleterious effects on teeth and their supporting structures. The number of medical textbooks and journal articles dealing with this subject is scant and can usually be measured in terms of sentences and paragraphs, not pages or volumes. Still, in the dental literature the body of data is growing as more and more studies are being conducted on the importance of smoking in the accumulation of plaque and calculus, the development of gingivitis and periodontal disease, and the state of oral health in general. It is an area of research that is just beginning and is destined to grow.

Among the dentally-related tobacco studies to date, there have been few clinical investigations on the effect of cigarette smoking on gingival crevicular fluid flow. A tedious undertaking in the past,

there now exists the technology to perform fluid flow measurements quickly and accurately.

Thus the primary purpose of this study is to evaluate the response of gingival crevicular fluid flow to chronic smoking. Additionally, it will attempt to add to the current literature regarding the prevalence of plaque and gingivitis in smokers, verify the reliability of chemical verification of tobacco consumption and a questionnaire that assesses nicotine dependence, and determine the relationship between these factors.

Review of the Literature

Tobacco and Periodontal Disease

The possible role of tobacco smoking in the initiation and progression of periodontal disease has been the subject of numerous investigations and clinical studies. Researchers have attempted to correlate plaque accumulation, calculus deposition, gingivitis, periodontal disease, and oral hygiene practices with smoking levels in order to determine what effect, if any, tobacco consumption contributes to these periodontal disease parameters. The dental literature on this subject yields few definite conclusions. Indeed the data are often conflicting and/or equivocal. This has been pointed out in reviews by Schwartz and Baumhammers,[1] Bastiaan and Reade,[2] and Bastiaan.[3]

Acute Necrotizing Ulcerative Gingivitis

Based on the results of a few studies, there does seem to be a strong, positive correlation between tobacco smoking and both acute necrotizing ulcerative gingivitis (ANUG) and calculus accumulation. In 1959 Bergeron[4] speculated that heavy tobacco consumption might predispose to the development of ANUG, but stopped short of declaring a direct cause and effect relationship. He did note, however, that many authors had mentioned this same observation. Decades later, Smeltzer[5] took exception to the claim of Bergeron and others by stating that out

of 100 of his patients with Vincent's infection, 37 percent did not smoke at all and that continued smoking had no effect on treatment success. However, Hirshfeld[6] observed in 1929 that during the period from 1910 to 1927 there was a concomitant 12-fold increase in cigarette consumption in the United States and an increased prevalence of Vincent's infection. He supported the relationship between the two phenomena by noting that women, heretofore relatively immune from the disease, were experiencing more frequent infections at the same time their smoking habit was becoming more fashionable. Finally, he offered anecdotal evidence that resistant cases of Vincent's infection responded to conventional therapy only when the patient stopped smoking. Stammers[7] concurred and suggested that the etiology might include both chronic mouth breathing in smokers and the irritating nature of the smoke itself. He also documented longer treatment times for patients with Vincent's infections who also smoked.

Pindborg[8,9] was the first to conduct studies that were specifically directed at the significance of tobacco smoking in the development of ANUG. In the first study[8] he examined 1,433 Danish Marines aged 16 to 28 years. He divided them into three groups based on their smoking practices (nonsmokers, those who smoked less than 10 grams per day, and those that smoked 10 grams or more per day) and into three groups based on gingival health (normal, chronic simple marginal gingivitis, and ulceromembranous gingivitis). While 1.5 per cent of nonsmokers had ulceromembranous gingivitis, 10.7 per cent of those subjects who smoked more than 10 grams of tobacco per day had the disease. Furthermore, 33 per cent of nonsmokers had normal gingiva compared to 22.3 per cent of

smokers. The harmful effects of tobacco were speculated to be caused by the irritating effects of tars, the heat of the smoke, the toxicity of carbon monoxide and/or other poisons, and/or the vasoconstrictive effects of nicotine on gingival blood vessels. In a second study involving 5,690 Marines, Pindborg[9] corroborated his earlier findings and showed that in the absence of local factors (calculus), the incidence of ulceromembranous gingivitis increased as the consumption of tobacco increased and that tobacco, by itself with all else being equal, affects gingival tissues in a detrimental manner. In a subsequent publication in 1951, Pindborg[10] drew the same conclusions.

Ludwick and Massler[11] found similar correlations between smoking and ANUG in a large group of Naval recruits. In 1952 they examined 2,577 male enlistees between the ages of 17 and 21 at the Great Lakes Naval Training Center and categorized them into five groups based on the number of cigarettes smoked per day (none, 5, 10, 15, or 20 or more). Although the incidence of ulceromembranous gingivitis was very low (only 20 cases), Pindborg's earlier observations were confirmed. Of these 20 persons, 13 smoked more than 16 cigarettes per day, six smoked from 6 to 15 cigarettes per day, and only one individual was a nonsmoker.

In 1983, Kowolik and Nisbet[12] demonstrated an almost invariable concurrence of smoking and ANUG. Of 100 patients with the disease, 98 were smokers. Despite the fact that over 85 per cent of the smoking group smoked more than 10 cigarettes daily, no correlation was found between smoking frequency and disease severity. It was also noted that a mean of 6-8 years was required for ANUG to develop following commencement of smoking. For this reason the authors concluded that

tobacco smoking is not a dominant factor in the etiology of ANUG, but only a consistent contributor.

Thus there is a consensus that cigarette smoking, while not the sole causative agent, is closely associated with the occurrence of ANUG. It is fair to state that most persons who suffer from ANUG do smoke and that their smoking habit is a contributory factor in their disease. It has been suggested that ANUG and smoking may represent two separate characteristics of the same emotionally stressed individual.

Calculus

Like ANUG, calculus has been consistently associated with tobacco consumption. Pindborg[8,9] observed that the presence of both supra- and subgingival calculus increased with increased use of tobacco. In these two studies, persons who were calculus-free dropped from 34.7 percent and 50.0 per cent of nonsmokers to 17.5 per cent and 30.9 per cent of smokers, respectively. Many years later Kowalski[13] used a more sensitive statistical analysis to evaluate Pindborg's data because the original method had failed to demonstrate which groups differed significantly. The new analysis showed that the probability of being calculus-free was greater for nonsmokers than for either group of smokers while the propensity for calculus formation among the two smoking groups was about the same. Additionally, Kowalski found subgingival calculus to be less affected by smoking tendencies.

Pindborg's findings have also been supported by a number of subsequent investigations involving groups of Norwegian soldiers,[14]

dental students,[15] Finnish Army recruits,[16] English and Irish industrial workers,[17] schoolchildren[18,19] and subjects who continued to smoke in a quit-smoking program.[20]

Using the Greene and Vermillion system,[21] Kristoffersen[14] scored calculus in a population of 321 Norwegian soldiers aged 19 to 23 years. After dividing the group into nonsmokers, smokers who smoked fewer than 10 cigarettes per day, and those who smoked 10 cigarettes or more per day, he found that there was a gradual increase in the calculus index as tobacco consumption rose, the correlation being statistically significant.

In partial fulfillment of the requirements for a Master of Dental Surgery degree from the University of London, Alexander[15] performed a study in a group of 200 dental students and 200 patients. Nonsmokers in both groups had significantly smaller supra- and subgingival calculus scores than smokers. One year later Ainamo[16] conducted a study in 167 Army recruits aged 18 to 26. On the basis of questionnaire responses, the population was divided into nonsmokers, light smokers (1-9 cigarettes per day), moderate smokers (10-20), and heavy smokers (more than 20). The Relative Calculus Index[22] increased linearly with increased tobacco consumption, and the difference between groups was significant.

Sheiham's[17] epidemiologic study in English and Irish laborers revealed similar findings. In both groups (a total of 2,119 subjects) nonsmokers had markedly less calculus than smokers according to the criteria of Greene and Vermillion.[21] In the Northern Ireland sample, people who smoked 1-10 cigarettes per day had cleaner mouths with less

calculus than those who smoked more. Studies done in children by Preber and Kant[18] and Modeer et al.[19] have found no differences in calculus indices, but it is important to note the low consumption and relatively short smoking histories in these groups.

In 1983 Feldman et al.[23] published the results of their study of 862 male volunteers from the Veterans Administration Dental Longitudinal Study. After the sample was divided into groups of nonsmokers, cigarette smokers, and pipe/cigar smokers and calculus accumulation was compared, cigarette smokers were found to have significantly more calculus than pipe/cigar smokers while both smoking groups had more calculus than nonsmokers. In an extensive review of data taken from the National Health and Nutrition Examination Survey, Ismail et al.[24] concluded that present smokers had significantly higher scores on calculus indices. The sample of 3,845 was divided into present smokers, past smokers, and those who never smoked.

Finally, in 1985 Christen et al.[20] conducted a longitudinal study to determine the oral effects of a chewing gum containing 2.0 mg nicotine used as an adjunct to a smoking cessation program. After an initial dental prophylaxis and a 15-week trial period on either the nicotine-containing or placebo gum, an incidental finding was a significant increase in calculus rates among those in both groups who continued to smoke. This study, as does the preponderance of other evidence, strongly suggests that smoking and calculus are closely associated.

Plaque

In sharp contrast to the almost invariably positive correlation between smoking and ANUG and smoking and calculus, conclusions regarding other types of periodontal disease and clinical indicators of periodontal disease are not as clearcut. When one examines other traditional measures of periodontal health, the consensus seen before is conspicuous by its absence.

Several studies have demonstrated increased levels of plaque in smokers. Sheiham,[17] using the Oral Hygiene Index,[21] found that English and Irish nonsmokers had markedly less debris than their smoking counterparts. Ainamo[16] used the Plaque Index of Silness and Löe[25] to score plaque in Finnish Army recruits. Plaque scores were lowest in nonsmokers, increasing with tobacco consumption to an almost statistically significant level. This accompanied a finding that moderate and heavy smokers tended to brush their teeth less often than nonsmokers.

Preber and Kant[18] found a trend towards greater amounts of plaque in 15-year old schoolchildren who smoked, but the differences did not approach significance. However, smokers were identified as those children who had smoked daily for at least six months irrespective of quantity. Average consumption and duration amounted in girls to five cigarettes a day for 1.2 years and in boys to seven cigarettes per day for 1.5 years. Although the differences were not significant, the study was a brief one and cigarette consumption was low.

In a similar study in a different group of schoolchildren, the number of cigarettes smoked was found to be a reliable predictor of plaque scores. Modeer et al.[19] studied a group of 232 Swedish schoolchildren whose mean age was 13.5 years. Children who smoked 10 or more cigarettes a day were classified as heavy smokers. Using the Plaque Index of Silness and Löe,[25] nonsmoking boys were found to have less plaque than those who smoked 1-9 cigarettes. The latter group had less plaque accumulation than boys who were heavy smokers. All differences were statistically significant and were not dependent on toothbrushing habits.

Preber et al.[26] studied the effects of cigarette smoking on the oral health of 134 Swedish Army conscripts. Regarding Plaque Index[25] scores, nonsmokers predominated in the low end of the frequency distribution, while smokers predominated in the high end. The differences were significant. The authors suggested that smoking may alter plaque adhesiveness in such a way to make it more difficult to remove.

In another study of short duration, plaque levels were only slightly increased in smokers. Bergstrom[27] measured plaque formation during separate five-day periods of smoking and no smoking, during which oral hygiene was discontinued. Plaque was scored every 24 hours at the same time each day. In this study smoking produced only a slight increase in early plaque formation. In the same comprehensive epidemiologic survey referenced previously, Ismail et al.,[24] after controlling for all other factors, concluded that smokers have significantly higher plaque scores than nonsmokers.

In 1984 Macgregor[28] reported the results of a study designed to determine whether there was any difference in plaque formation between 64 habitual smokers and 64 nonsmokers, and to determine if such a difference, if it existed, might be due to variations in unsupervised but observed toothbrushing patterns. Smokers had significantly more plaque, but the difference could not be linked to a disparity in toothbrushing frequency.

Despite the weight of the evidence from the aforementioned studies, almost as many investigators have found no statistically significant differences in plaque accumulation between smokers and nonsmokers.

Kristoffersen[14] studied periodontal conditions in 321 soldiers at two Norwegian military camps. The system of Greene and Vermillion[21] was used to assess oral hygiene. The sample population was divided into three groups as previously outlined. Although there was a gradual increase in scores of debris, calculus, and oral hygiene indices with increasing consumption of tobacco, the associations between smoking and plaque scores for all three groups were not significantly different.

Alexander[15] reported the same observation when he examined a group of 200 dental students and 200 dental patients. Even though nonsmokers had significantly less calculus, there was no difference in bacterial plaque between them and the smoking group.

Bastiaan and Waite[29] in 1978 conducted a study in which the rate of plaque formation in 10 smokers and 10 nonsmokers was assessed during a 10-day period of suspended oral hygiene. Plaque indices were recorded on days 3, 7, and 10. There was a trend for more plaque accumulation with time in subjects who smoked 10 cigarettes or more daily, but at

none of the time intervals were the differences between the two groups significant.

In a study conducted in a private periodontal practice, Swenson[30] was unable to detect a difference in plaque scores between 297 smokers and 258 nonsmokers. The sample was divided into five groups: nonsmokers and those who smoked an average of 10, 20, 30, and 40 or more cigarettes daily. On day 1 plaque scores were recorded using the method of O'Leary et al.[31] Patients were dismissed for 6 to 8 days and then rescored. Differences were so small that a statistical analysis was deemed unnecessary.

Macgregor et al.[32] assessed plaque formation in two different manners. After two separate experiments in which a 48-hour hygiene-free period was preceded by either unsupervised toothbrushing or a professional prophylaxis, plaque was first measured in smokers and nonsmokers with the Plaque Index (PlI) of Silness and Löe.[25] Then plaque was collected from all tooth surfaces and weighed. The mean PlI and wet weight were marginally higher in smokers in both studies, but the difference was not significant.

Finally Christen et al.[20] noted slightly higher plaque scores in all participants in a quit-smoking program. Although increases from baseline were numerically smaller in those participants who stopped smoking during the study period, the differences were not statistically significant. Neither continued smoking nor the cessation of smoking seemed to affect plaque scores.

To further cloud the issue, in 1983 Feldman et al.[23] reported that cigarette smokers had less plaque than pipe and cigar smokers, and that

both groups of smokers had less plaque than nonsmokers. The oral hygiene practices of the 862 participants were unknown, but the authors postulated that an increased salivary flow in smokers may explain the effect on plaque formation.

In addition to those studies evaluating plaque formation per se, several have examined related factors. Kenney and associates[33] in 1975 evaluated the effect of smoking on intraoral oxidation-reduction potential (Eh) levels and intraoral pH in 38 male dental students, 19 of whom were smokers. The development of plaque is associated with a fall in Eh which shifts the ratio of anaerobes to aerobes in plaque in favor of the former. That is, anaerobic bacteria increase in numbers. After an initial dental prophylaxis and 14 days of no oral hygiene, there were no differences between smokers and nonsmokers in resting Eh, the population of anaerobic bacteria in plaque, or oral pH values.

Colman et al.[34] compared the microflora in different parts of the oral cavity (tongue, hard palate, lingual and proximal areas of the mandibular first bicuspid and cuspid, mandibular left incisors, mandibular right incisors, and mandibular right cuspid and bicuspid) in five smokers and four nonsmokers. The smoking group consumed 20 or more cigarettes per day. Significant differences were found in two sites. Smokers had fewer neisseria and more bacteroides and veillonella on the tongue, while the palatal mucosa harbored fewer neisseria. The authors reasoned that tobacco smoke may be selectively toxic to neisseriae, in addition to producing anaerobic conditions under which neisseria do not fare well.

Kenney et al.[35] used saline rinses to harvest oral polymorphonuclear leukocytes (PMN) from smokers and nonsmokers and measured their ability to phagocytize latex spheres and to exclude trypan blue. PMN taken from smokers were less able to perform either task. In addition, smoking one cigarette immediately prior to cell collection impaired PMN function in both smokers and nonsmokers. Impairment of PMN could contribute to periodontal disease through reduced defenses via bacterial plaque accumulation. Earlier, Eichel and Shahrik[36] reported essentially the same findings, which they attributed to a decrease in oxygen consumption by these cells.

Bastiaan and Waite[29] found a significantly higher proportion of gram-negative to gram-positive organisms on the third day of plaque development in smokers, but at days seven and ten the differences became non-significant and in fact the percentages became similar. They postulated that the difference in staining characteristics could have been due to an alteration in oxidation-reduction potential, but deferred further comment until more research could be conducted.

Gingivitis

Much the same situation exists regarding the relationship between gingivitis and smoking. Gingivitis here is defined as "inflammation of the gingiva" characterized by "soggy puffiness that pits on pressure, softness and friability, ready fragmentation on probing, and pinpoint areas of redness and desquamation."[37] Various studies have found that

smokers have an increased incidence of gingivitis, while others have found just the opposite. Herulf[38] examined 535 dental students and found that gingival changes were far more prevalent in smokers. On the basis of their clinical study in 1346 factory workers, Arno et al.[39] concluded that the effect of tobacco as an etiologic factor in periodontal disease was strongly substantiated. The employees were classified into four groups: nonsmokers, small consumption (1-9), medium consumption (10-20), and high consumption (greater than 20 cigarettes per day). When hygiene and age were kept constant, there was a significant correlation between tobacco consumption and gingivitis, although smoking was not considered to be the most significant factor in the etiology of periodontal disease.

In 1970 Alexander[15] studied the relationship between tobacco smoking and periodontal health in 200 dental students and 200 dental patients. Findings regarding calculus and plaque have already been described. Based on assessments of the free margin, the papilla, and the attached gingiva, he found that student nonsmokers had a lower, but not statistically different mean gingival inflammation score. However, in the patient group the difference was significant, with nonsmokers again displaying healthier gingiva.

Preber and Kant[18] in 1973 reported that even in 15-year-old schoolchildren, persons with lower Gingival Index[40] scores tended to be nonsmokers. These same investigators, together with Bergstrom,[26] used the same Gingival Index of Löe and Silness[40] and found a statistically higher level of gingivitis in young Army personnel who smoked. However, this difference disappeared when plaque levels were considered.

In 1985 Christen and associates[20] published observed effects of a nicotine-containing chewing gum on oral tissues. Although the gum had no effect on gingivitis, subjects who continued to smoke had a statistically higher level of gingivitis than those who quit smoking. Smoking, regardless of the gum used (nicotine-containing or placebo), exerted significant effects upon gingival health.

However, other studies have not been able to detect differences in gingivitis between smokers and nonsmokers. Pindborg's early studies[8,9] showed no differences in the incidence of "chronic simple marginal gingivitis" in the two groups. Ludwick and Massler[11] were unable to correlate the number of inflamed areas (PMA[41]) with the number of cigarettes smoked per day. Kristoffersen[14] could not find a significant association between smoking and scores of Russell's periodontal index[42] in Norwegian soldiers.

In 1971 Ainamo[16] found no corresponding increase of the mean Gingival Index[40] scores as smoking increased. Identical scores were recorded in nonsmokers, light smokers, and moderate smokers. Bastiaan and Waite[29] found only slight variations in Gingival Index[40] between smokers and nonsmokers who participated in a 10-day experimental gingivitis study. A recent study by Markkanen and co-workers[43] was done in a Finnish population of individuals aged 30 years and older. There were only small differences between smokers and nonsmokers regarding Gingival Index[40] and pocket depth, although it was noted that nonsmokers had less severe periodontal disease.

Plaque vs Gingivitis

Interestingly, when the incidences of plaque and gingivitis are compared in the same study, a paradox emerges. For example, Alexander[15] reported an increased incidence of gingivitis in smokers, but no difference in plaque scores between the same group of smokers and a group of nonsmokers. Ainamo[16] reported just the opposite: a trend towards more plaque in smokers but no difference in gingivitis. He attributed this apparent dichotomy by speculating that the heat and toxins of the smoke may cause hyperkeratinization of the gingiva or may reduce bacterial virulence. Finally Feldman[23] found equivalent levels of gingivitis in cigarette smokers, pipe/cigar smokers, and nonsmokers although both smoking groups had less plaque than the nonsmokers.

Bone Loss, Pocket Depth, and Periodontal Disease

In studies that have reported bone loss and pocket depth, both tend to be greater in smokers. Herulf[38] used radiographs of the mandibular anterior teeth and observed that interdental bone height was significantly lower in smokers than nonsmokers. However, plaque accumulations were not part of the assessment. Arno and others[44] evaluated the influence of tobacco consumption on the speed of alveolar bone resorption. A sample of 728 male industrial workers was divided into four groups according to smoking habits using the criteria of Arno

et al.,[39] and into five groups according to age. Oral hygiene was classified as good, medium, or not good. Ten radiographs were taken on each subject, and bone loss was measured as a percentage of maximum bone height. Allowing for age variations, a "systematic" correlation was found between bone loss and tobacco consumption, suggesting that tobacco is a complicating factor in periodontal disease that may accelerate bone destruction when local and systemic factors are present.

Feldman et al.[23] reported that cigarette smokers had significantly greater pocket depths than nonsmokers and greater bone loss than either pipe smokers or nonsmokers. This was in spite of the fact that all smokers had less plaque and gingival inflammation than nonsmokers.

Conversely, Preber et al.[26] found no association between smoking and pocket depths or bone loss. Periodontal pockets were measured with a probe; bone loss was assessed by projecting radiographs magnified 10 times onto a screen with horizontal lines at five millimeter intervals. They noted, however, that bone loss is seldom pronounced in a population whose mean age is 21.9 years. They also observed that small changes in bone density may be undetectable by conventional radiographs.

Several studies have found that smokers have a higher percentage of periodontal disease[45,46] and/or more severe[17,46] forms of the disease. In a study of 206 Norwegian Army recruits, Brandtzaeg and Jamison[45] in 1964 reported a trend toward higher plaque, debris, calculus, and oral hygiene indices with increased tobacco consumption. Soloman and co workers[46] reviewed the results of 9561 dental examinations performed between 1957 and 1965, focusing on reports of gingival recession and alveolar bone loss. When those results were compared with smoking

histories obtained from personal interviews, the prevalence of periodontal disease was higher for both men and women. In younger groups, disease prevalence in women who smoked resembled that seen in older male smokers. In older groups, disease prevalence in female smokers was closer to that of nonsmokers. This was attributed to the observation that older women had smoked less than men and younger women. It was concluded that women aged 20 to 39 years, and men aged 30 to 59 years, have twice the risk of periodontal disease if they smoke.

In a study at the University of Michigan, Summers and Oberman[47] studied the association between periodontal disease and 12 selected variables, among them cigarette smoking. A total of 324 subjects were examined and gingival disease was assessed with the periodontal disease index (PDI) described by Ramfjord.[48] Smoking levels were determined via medical histories. In all age groups for both sexes, smokers had numerically higher PDI scores than nonsmokers. This difference was significant in two sub-populations: males aged 40 to 59 and males 50 years and older. The percentage of male smokers, as well as the mean amount of cigarettes smoked by males, was higher than the like categories in females. These two factors may have accounted for the lack of differences between female smokers and female nonsmokers.

Two recently published studies support the view that smoking and periodontal disease are associated. In 1986 Preber and Bergstrom[49] reported that compared to nonsmokers, smokers had twice the chance of developing periodontitis. Bergstrom and Eliasson[50] found that bone loss was accelerated in smokers and that regardless of good oral hygiene, smoking exerted a negative influence on periodontal health.

Although Sheiham[17] reported in 1971 a greater severity of periodontal disease in smokers, when allowing for similar levels of oral hygiene the differences were not statistically significant. Lilienthal[51] found no relationship between smoking and periodontal disease in a study of 854 subjects in a private dental practice in Australia. Only a qualitative assessment of smoking was made, i.e., whether a person smoked regularly or not. On the other hand, Ismail et al.[24] found that the association between poorer levels of periodontal health and smoking remained even after accounting for age, sex, race, oral hygiene, socio-economic status, and frequency of toothbrushing.

Miscellaneous Parameters

Various articles have dealt with a wide range of the potential effects of tobacco on oral tissues that do not fall into the categories of ANUG, calculus, plaque, gingivitis, bone loss, pocket depths, or periodontal disease per se. They do, however, relate to oral health in general and are mentioned here for the sake of completeness and because selected studies focus on peripheral phenomena that relate directly or indirectly to the oral disease process.

In 1968 Shuler[52] published the results of a study designed to measure the local effect of cigarette smoking on the blood circulation of the oral mucosa. When nicotine, for example, is absorbed into the blood vessels of the oral mucosa, noradrenalin is secreted and vasoconstriction results.[52] These actions could have a detrimental

influence on gingival health. In this particular study, nonsmokers and regular smokers who had been deprived of cigarettes for 48 hours, smoking one cigarette decreased oral mucosa blood flow by 50% and 60% to 70%, respectively. There was no effect on those subjects who had been allowed to smoke prior to the experiment, indicating that whatever vasoconstriction was to occur had already taken place.

In 1970 McKendrick et al.[53] reported that smokers have almost twice as much staining as nonsmokers. In a review of the oral effects of tobacco, Christen[54] included such things as hairy tongue, altered taste and smell, periodontal disease, abrasion and discoloration of the teeth, delayed wound healing, sinusitis, leukoplakia, and cancer.

Sweet and Butler[55] in 1978 reported an increased incidence of localized osteitis following the removal of mandibular third molars in patients who smoked during the postoperative period.

Bennet and Reade[56] measured salivary immunoglobulin A (sIgA) levels in nonsmokers and two groups of smokers (those who had smoked in excess of 20 cigarettes a day for 20 years and 40 years). There were no differences between nonsmokers and the 20-year smokers, but SIgA concentrations in the 40-year smokers were significantly depressed. This reduction may have been due to an immunosuppressive effect of the combustion products of tobacco, which may in turn have fostered intraoral neoplastic changes in the smoker. Olson et al.[57] found no differences in sIga between nonsmokers and smokers who used either a nicotine-containing or a placebo gum in a quit-smoking effort.

Some studies have investigated gingival bleeding tendencies. Bergstrom and Floderus-Myrhed[58] reported that gingival bleeding

propensity was less prevalent in twins with a high lifetime exposure to cigarette smoking than in their twin partners with low lifetime exposure, or none. Interestingly, the smoking twin had more alveolar bone loss and more missing teeth. It was pointed out that bleeding tendencies were based on self-report and not clinical indices. In contrast, a second study evaluated gingival bleeding by counting the number of sites that bled on probing as a percentage of the total. Preber and Bergstrom[59] examined 10 nonsmokers and 10 smokers with a 15-year, 20-cigarette per day history. Although the smoking group had significantly more plaque than nonsmokers, their bleeding tendency was reduced. This was later reported in another study by Preber and Bergstrom.[49] The vasoconstrictive action of nicotine and other tobacco smoke constituents was offered as an explanation for the apparent contradiction.

Summary

The relationship between cigarette smoking and periodontal disease falls into two categories. Calculus and ANUG have been shown to be consistently correlated with tobacco consumption. On the other hand, studies involving the assessment of plaque and gingivitis in smokers and nonsmokers are conflicting. While it seems fair to state that smoking and periodontal disease are strongly associated, the question remains as to whether there is a cause and effect relationship. There have been no controlled studies in humans to substantiate this claim, and there is

not likely to be one for ethical reasons. However, epidemiologic studies like the one conducted by Ismail et al.[24] come as close as is possible and have demonstrated that smoking has an adverse effect on periodontal health that cannot be totally explained by differences in oral hygiene status.

Nevertheless, much of the inconsistency and confusion surrounding this controversial issue stems from the wide variation in criteria used for disease assessment in the studies just reviewed. These criteria have ranged from personal observation to reviews of dental records to a variety of similar but not identical indices, all of which have an inherent subjectivity. This non-standardization makes a direct comparison of two or more studies difficult.

A paucity of information exists regarding possible differences in gingival crevicular fluid (GCF) flow in smokers and nonsmokers. A review of that subject is now in order.

Gingival Crevicular Fluid

Flow of tissue fluid through the crevicular epithelium has been the subject of considerable research since the 1950s when Waerhaug noted that foreign matter (India ink) introduced into gingival pockets was eliminated in 48 hours,[60] and that saturated dyes placed at the entrance would not diffuse into the pocket.[61] The latter observation was supported by Brill[62] using charcoal particles and Harvey[63] using silver alloy particles, both indicating the existence of a physiologic flow of

fluid from the crevice. Initially the source of gingival crevicular fluid was questioned in that there was doubt as to whether the crevicular epithelium was permeable to small molecules. Brill and Krasse[64] and Brill and Björn[65] helped to resolve those doubts by demonstrating that fluorescein sodium, injected intravenously in dogs or administered orally in humans, could be detected on paper strips inserted into the gingival crevice, but not on strips placed on other epithelial surfaces. Fluorescein sodium is taken up by blood and tissue fluids, but will not penetrate intact epithelium. These early studies demonstrated that fluid did pass from deeper tissues, possibly capillaries, through the crevicular epithelium, although the significance of this finding was unclear.

In addition to verifying the permeability of crevicular epithelium, Brill and Björn[65] noted a correlation between the amount of fluorescein collected on paper strips (i.e., fluid flow) and the severity of inflammation. The gingival health of the 12 subjects in their study ranged from normal gingiva to generalized chronic gingivitis. Whether their observations indicated a physiologic or pathologic condition could not be established, but the authors concluded by stating that "inflamed epithelium yielded greater amounts of fluorescent fluid than did healthy gingiva." They suggested, as did Waerhaug,[60,61] that the stream of fluid coming from the sulcus may be a self-defense mechanism that flushes particulate matter out of and/or blocks its entrance into the sulcus. In another study in dogs, Brill and Krasse[66] demonstrated that mechanical stimulation such as chewing and toothbrushing can increase GCF flow, but that this flow returns to baseline within ten minutes.

After a subsequent confirmatory study in dogs, in which gingivitis was experimentally induced by varying the consistency of the animals' diet, Brill[67] found that a marked production of fluid was associated with an extensive inflammatory reaction (as determined histologically by the occurrence of rete pegs and an increase in the number of inflammatory cells) and vice versa. On the basis of these findings, he suggested that a similar filter paper diagnostic test could be used to evaluate the progress of periodontal therapy in humans.

In a clinical study involving 307 observation sites in 27 human subjects, Mann[68] used a modified fluorescein sodium solution/filter paper technique (although strips were still placed as deeply as possible until resistance was felt), as well as a microscopic analysis of the wetted area, to correlate fluid flow per unit area with both pocket depth and gingival scores. The analysis showed that inflammation had a stronger relationship to the amount of fluid than did pocket depth. By assessing the ratio of sodium to potassium in gingival fluid and comparing that ratio with extracellular tissue fluid, Krasse and Egelberg[69] further suggested that gingival fluid represented an inflammatory exudate rather than a transudate because of the increase in intracellularly-derived potassium, as seen in metabolically altered tissue. Mann's study lent credence to this hypothesis by noting that there was minimal flow in healthy gingiva and maximum flow in conditions of inflammation.

Egelberg[70] also found a highly significant correlation between the amount of gingival fluid and the clinical estimate of the degree of inflammation. Likewise, Björn et al.,[71] in a study of 170 humans, found

a highly significant correlation between the amounts of gingival exudate and the Gingival Index[40] scores. They found fluid even in gingival areas that were deemed to be clinically free of inflammation.

Löe and Holm-Pedersen[72] conducted a study involving 336 sites in 118 adult humans. Using both an extracrevicular and intracrevicular technique for filter paper placement, gingival fluid was collected from each site. The extracrevicular method consisted of adapting a strip to the attached gingiva and tooth surface, thus bridging the entrance to the crevice. In the intracrevicular method, care was taken to place strips only at the entrance to the crevice in order to avoid false positives. After removal, the strips were stained with a 0.2 per cent solution of ninhydrin and the width of the stained area was measured to the nearest 0.05 mm with a magnifying glass. Investigations were carried out in normal gingiva (resting and mechanically stimulated), in clinically inflamed gingiva, and in gingiva following intentional withdrawl of oral hygiene (experimental gingivitis as described by Löe et al.[73]). The results confirmed those of Mann.[68] Crevices of normal gingiva, whether resting or stimulated, yielded no fluid flow. Crevices from the other two groups showed the presence of fluid, the amount of which varied according to the severity of the inflammation. In the experimental gingivitis group, this flow diminished a few days after gingival inflammation was reversed by resumption of oral hygiene. The investigators noted that the flow routinely began before there was clinical evidence of gingivitis, suggesting that GCF flow is an early sign of inflammation, is in fact an inflammatory exudate, and that it could be used as a diagnostic tool to assess the subclinical state of

the gingiva. The results of this study conflict with those of Brill and Björn[65] in that the latter study found gingival fluid under all conditions, whereas the former found none in healthy gingiva. The difference was attributed to the manner in which strips were inserted into the crevice, i.e., the method used by Brill and Björn[65] may have amounted to minor trauma.

Arnold et al.[74] investigated the variations in crevicular fluid flow before but, more important, after gingival surgery, and especially during the healing period. In the preoperative evaluation phase, all patients who presented with periodontal disease were biopsied and GCF flow was measured. In all cases biopsy specimens revealed chronic inflammation and all patients exhibited marked fluid flow. During the healing period, the initial rise and subsequent decline in histologic, cytologic and clinical inflammation were mirrored by an initial increase and then fall in GCF flow rate. The authors concluded that a direct relationship existed between gingival healing and fluid flow.

Results of a related study by Sandalli and Wade[75] confirmed those of Arnold et al.[74] and differed only slightly in the magnitude of flow reduction due to the fact that all surgical procedures (gingivectomies and flap procedures) were preceded by an initial preparation phase that reduced baseline flow measurements. An additional finding was a high correlation between pocket depth and the amount of gingival fluid.

In a series of investigations on the permeability of dento-gingival blood vessels in dogs, Egelberg[76,77] showed that gingival fluid flow could be stimulated in healthy gingiva by topical application of histamine and by massaging the gingiva and/or scraping the gingival

crevice with a blunt instrument.[76] But in clinically healthy gingiva that are not stimulated, abnormal permeability of the gingival vessels does not occur, as evidenced by the failure to collect fluid on 90% of the paper strips placed in the orifice of crevices.[77] These results are in accordance with the findings of Löe and Holm-Pedersen,[72] but conflict with those of Brill and Krasse[64] and Egelberg.[70] Again, the discrepancy was attributed to different collection techniques. Also the smaller fluorescein molecule, used as a tracer by Brill and Krasse,[64] may be better able to permeate the crevicular epithelium than the proteins assessed in Egelberg's series of studies.

In addition to clinical gingivitis, other factors have been shown to affect gingival fluid flow. They include regular use of oral contraceptives,[78] menstruation[79] (but only if menstruation is preceded by a state of gingival inflammation[80]), and progesterone administration.[81] On the other hand, Holm-Pedersen and Löe[80] found that pregnancy and the period immediately post-partum did not seem to affect gingival fluid flow per se. In 1967 Bissada[82] determined that GCF flow follows a circadian rhythm with the highest flow rate occurring at about 2200 hours (10 P.M.), four hours after the peak in body temperature. However there were variations between individuals and between different crevices in the same individual.

In 1969 Oliver et al.[83] conducted a study of 60 labial and buccal gingival areas in 53 patients in an effort to determine the relationship between GCF flow, a gingival index, and a histologic examination of the same tissue. An intracrevicular method was used for placement of filter strips, after which they were stained with a solution of 0.2% percent

ninhydrin. The stained areas were then measured with a magnifying glass. Gingival Index (GI)[40] scores and the amount of GCF were strongly correlated. Biopsies, in which inflammatory cell density was determined, correlated well with both GI scores and GCF flow, although more closely with GI scores. The earlier findings of little or no exudate in clinically disease-free gingiva were confirmed.

Rudin et al.[84] in 1970 conducted a similar study of 13 patients in which they measured GCF flow from 30 teeth and compared the results to pocket depth, clinical inflammation, and microscopic examinations with respect to inflammatory cells, collagen fibers, interstitial connective tissue and blood vessels. Sulcular fluid flow increased with increasing inflammation and correlated with round cell infiltration, but did not correlate with the number of connective tissue cells or blood vessels. Again, in healthy marginal gingiva, only traces of GCF were measured. The authors stressed the importance of placing the paper strip precisely at the entrance of the sulcus and supported the view that GCF measurements reflect the severity of gingivitis.

In 1977 Borden et al.[85] reported the results of a study to determine if crevicular fluid flow is indicative of the severity of inflammation when age and sex are considered. In 120 subjects, 60 males and 60 females, they found that neither age nor sex affected the relationship between gingivitis and fluid flow. Their results also confirmed earlier claims that measuring fluid flow is a sensitive, objective technique for assessing gingival health.

In a study involving 48 adult patients, Engelberger et al.[86] evaluated 95 interdental sites. Obvious and highly significant

correlations were demonstrated between gingival fluid flow and a sulcular bleeding index ($r=0.597$, $P<0.001$) and a papillary bleeding index ($r=0.622$, $P<0.001$). Bleeding on probing was considered to be the first clinical sign of gingivitis.

Not all clinical investigations of GCF flow are in agreement with the aforementioned studies. Orban and Stallard,[87] using the same basic technique as Oliver et al.,[83] conducted a study among dental patients selected at random. GCF measurements and plaque (Ramfjord's criteria[48]) were compared to the results of gingival biopsies which were scored according to the amount and extent of inflammatory exudate. Scatter diagrams showed that GCF measurements did not directly relate to biopsy scores, while plaque scores were a better predictor.

In a study of 48 university students, Wilson and McHugh[88] compared what they termed a Gingival Exudate Index with gingivitis indices (modified versions of the PMA Index of Massler and Schour[41] and the Gingival Index of Löe and Silness[40]) and a plaque index (a modification of the Oral Debris Index of Greene and Vermillion[21]). Strips were stained with 0.2% ninhydrin and examined under magnification. Scatter diagrams indicated a poor correlation between the Gingival Exudate Index and all other indices for the whole mouth, but did correlate well with the Gingival Index for individual gingival surface areas. In addition, very few of the areas that were scored clinically as normal did not yield GCF, thus failing to confirm the results of earlier studies.

In 1976 Daneshmand and Wade[89] conducted a clinical trial in 30 subjects with varying degrees of periodontal disease. A Gingival Index[40] was recorded, GCF flow was measured according to the method

outlined by Loe and Holm-Pedersen,[72] and biopsies were taken. Low positive correlations (r=0.3) were found between histologic indices (extent of inflammatory cell infiltration and the number of extravasated inflammatory cells) and GCF. A moderate correlation was found between GCF and GI scores.

An extensive discussion of crevicular fluid, its origin, function, and composition, as well as its significance in regard to periodontal health, can be found in two extensive works by Cimasoni.[90,91]

Three distinct methods using absorbent filter paper strips have been employed to measure gingival fluid. Some studies[64,72] have used an extracrevicular technique in which a filter paper strip is adapted to the surface of the attached gingiva and the adjacent tooth, bridging the opening to the gingival crevice. By far the majority of studies have used an intracrevicular method, of which there are two. Some investigators have inserted paper strips deep into the crevice until resistance is felt. Because of the possibility that this technique might induce trauma and artificially stimulate gingival fluid flow, many more workers have elected to place the end of the strip just inside or precisely at the entrance to the crevice. The decision as to which intracrevicular technique to use seems to have been an arbitrary one. Egelberg and Attström[92] compared the two methods in a clinical study and found that although the orifice method showed less variation between samples, both techniques were acceptable. They also supported the concept that gingival fluid measurement is a sensitive indicator of gingival health. Differences in fluid amounts were evident one day after cessation of oral hygiene.

Several methods have also been used to quantitate the amount of fluid collected and have been summarized in reviews of the literature by Golub and Kleinberg,[93] Abbott and Caffesse,[94] and Smith.[95] Kaslick and associates[96] used calibrated microcapillary tubes not only to collect but also to directly measure the amount of fluid. However, this method requires large volumes of fluid and extended periods of time to achieve accuracy.[93] Weinstein et al.[97] used pre-weighed twisted thread to collect fluid, but sample evaporation made measurement difficult.[93] The use of filter paper strips, by far, is the most popular procedure. In early studies[64-68,82] strips were used to collect fluorescein sodium and then photographed under ultraviolet light. Later, the wetted area was stained with 0.2 percent ninhydrin, which has an affinity for free alpha-amino groups on amino acids in the sample, and the stained area was measured under high power magnification.[86-88,98,99]

In recent years a device called the Periotron (Harco Medical Electronics, Inc., Irvine, CA) has been marketed that electronically measures and quantitates the amount of fluid on a paper strip. The Periotron is said to remove the subjectivity of the other methods and overcome the problem of sample evaporation.[93,94]

The Periotron has a metering system in which two "jaws" function like plates in a condenser. If a dry strip is inserted between the jaws, the capacitance is at its maximum and the electronic circuitry registers a zero on the readout screen. When a wet strip is inserted, the capacitance drops and the readout increases in direct proportion to the area wetted, thus quantifying the amount of fluid collected.

Although the technique is relatively new, several laboratory and clinical trials have evaluated the Periotron, and many more studies have used the device to measure periodontal health in response to other clinical therapies. Suppipat and Suppipat[100] did an *in-vitro* study in which they tested an early version of the Periotron, the HAR-600 Gingival Crevice Fluid Meter (GCFM). They found that the position of the strip placed between the jaws of the instrument, the viscosity and ionic strength of the fluid measured, and the temperature and humidity of the environment could have an affect on the meter readings. Generally, readings tended to be higher when the filter paper strip was placed between the front halves of the jaws, when the viscosity was higher, when the ionic strength was lower, and when the temperature or the humidity was increased. Despite the variability, there was a linear relationship between the HAR-600 GCFM and the traditional ninhydrin-staining technique.

Jameson[101] used the HAR-600 GCFM to compare the volume of crevicular fluid from teeth restored with full coverage with subgingival margins and nonrestored teeth. The fluid volumes from the restored teeth were twice the values obtained from contralateral unrestored teeth, the difference being highly significant. This device has also been used to compare the effects of mechanical tooth cleaning and chlorhexidine mouthrinses on gingival fluid flow after gingivectomy.[102] Results showed the expected initial increase in flow from day 0 to day 14, followed by a decrease. Although the readings varied 15% daily due to environmental conditions, the error was considered acceptable and the measurements accurate.

Suppipat et al.[103] used the HAR-600 GCFM to confirm or deny Bissada's[82] earlier circadian rhythm finding. They found that "time of day" did not influence the rate of gingival fluid flow. This device was also used by Renner and others[104] to chart the health status of overdenture abutments from initial periodontal therapy through denture insertion, by Stoller et al.[105] to evaluate the efficacy of an amine fluoride mouthrinse in reducing gingival inflamation, by Ringleberg and others[106] to compare the gingival health in diabetic and non-diabetic children, and by Biswas and associates[107] to study the effect of age and sex on fluid volume in adolescents.

Tsuchida and Hara[108] used the HAR-600 GCFM along with Gingival Index[40] scores, pocket depths, and an assay for acid phosphatase content of the collected fluid to evaluate the effect of initial preparation procedures in 10 subjects with periodontitis. Comparisons were made between GCF flow and all other test parameters. All of the mean Gingival Index scores, pocket depths and HAR-600 GCFM readings decreased postoperatively, the results being statistically significant. In addition, when fluid measurements from the HAR-600 GCFM were compared with Gingival Index, pocket depths, and acid phosphatase levels, all were highly correlated ($r=0.65$, 0.74, and 0.92, respectively). Despite the findings of Suppipat and Suppipat,[100] no problems were encountered regarding temperature and humidity. The authors felt that "while other clinical examinations using inspection or palpation may be subjective, the gingival fluid measurement (used in this study) is a sensitive and quantitative method."

In a combination in-vitro/in-vivo study, Garnick et al.[109] evaluated a second-generation device, the Periotron 600. They found that substances having different dissipation constants can result in variations of volume assessment. For example, equal volumes of fluid from different subjects showed statistical differences. However, when Gingival Index[40] scores were compared with Periotron readings, there was a direct linear relationship and a high correlation (r=0.8260). The authors stated that the Periotron 600 may be appropriate for longitudinal studies of gingival fluid, but not for comparisons between individuals.

A more elaborate clinical investigation was done by Shapiro et al.[110] in which Gingival Index[40] scores, Periotron 600 readings, and biopsy results in 45 subjects were compared. The Gingival Index and the Periotron 600 readings were closely correlated, but the histologic examination demonstrated no correlation with either of the other two parameters. Golub et al.[111] also showed that a direct relationship existed between gingival flow as measured with the Periotron and the Gingival Index.[40]

Kowashi and associates[112] used the Periotron 600 to compare gingival fluid flow with the concentration of polymorphonuclear leukocytes (PMNs) in gingival washings during a 21-day period of experimentally-induced gingivitis. The two variables did not correlate with each other, but the results confirmed that the rate of gingival fluid flow is the more reliable indicator of gingival health. Also, a much greater variability was found for the PMN concentration than for the amount of fluid.

In another clinical study, Wunderlich et al.[113] used Periotron 600 measurements and bleeding tendencies to evaluate the effects of waxed and unwaxed floss on gingival health. Although fluid flow (and bleeding) were least with waxed floss at the end of 56 days, the differences were not significant, the range being less than one Periotron unit based on a scale of 0 to 200. In their study of protein concentration in gingival crevicular fluid, Hattingh and Ho[114] first measured flow rates with the Periotron 600. Maltais and Messer[115] used the Periotron 600 to compare GCF and traditional periodontal indices in children and again showed that GCF flow correlated significantly with increasing scores of gingivitis, plaque, and crevice depth.

Despite the favorable impressions regarding the instrument's usefulness expressed in these investigations, two studies[116,117] found the Periotron 600 to be less accurate than the traditional ninhydrin staining method. Nevertheless, when Taggart et al.[118] compared the accuracy of the Periotron 600 with estimations of fluid flow made by the ninhydrin staining technique under clinical conditions, they found both to be statistically equivalent. Furthermore they noted that the Periotron, unlike the staining method, provided immediate results. Also, as stated in other studies, GCF correlated well with periodontal indices.

A third generation device promising greater accuracy, the Periotron 6000, was introduced in 1983. There have been three <u>in-vitro</u> studies involving this model. In the first, Hinrichs et al.[119] compared the variability of the Periotron 6000, the Periotron 600, and the traditional ninhydrin staining method. Known quantities of normal human

serum were delivered to filter paper strips with a syringe. The procedure was repeated using distilled water containing 0.1 per cent methyl green, but only for the two Periotron units. Fluid volumes were then measured. Coefficients of variation for the Periotron 6000 were significantly smaller than those of the Periotron 600 or the ninhydrin method. Therefore, it was concluded that less variability does indeed exist in measurements taken with the Periotron 6000 and that 2.25 to 6.25 times as many samples must be measured with the other two methods to obtain the same level of precision.

The second study by Bickel and Cimasoni[120] tested the ability of the Periotron 6000 to measure known volumes of different fluids and solutions, among them crevicular fluid. The device was shown to be very accurate in estimating the small quantities of fluid characteristic of GCF. The coefficient of correlation for fluid amounts ranging from 0 to 400 nl was 0.97. Furthermore, confidence limits were narrow for crevicular fluid, and there was a linear relationship between Periotron readings and fluid volumes. The results were interpreted to signify that the Periotron 6000 is a highly reliable device to study GCF in clinical studies.

The third <u>in-vitro</u> study again compared the Periotron 6000, the Periotron 600, and the ninhydrin staining method. Hinrichs and others[121] assessed the ability of all three methods to measure distilled water, saline and normal human serum both quantitatively and qualitatively. The latter determination is most important in investigations comparing gingival flow in many subjects. All three systems demonstrated a linear relationship between fluid volumes and the

numerical values assigned. The Periotron 600 was found to be more sensitive to qualitative differences in fluids than the other two methods. However, the Periotron 6000 was relatively insensitive to these differences, with the ninhydrin method slightly more sensitive than the Periotron 6000. The authors concluded the article by stating, "If one is concerned about the impact of qualitative differences in fluids upon the quantitative measurements, the Periotron 6000 or the ninhydrin method appear to be the systems of choice." This would seem to suggest that the Periotron 6000 is well suited for epidemiologic studies in which GCF flow is compared across many subjects. According to Kleinberg and Golub,[122] the Periotron has largely superseded other methods of measuring fluid flow and is able to accurately detect as little as five nanoliters of fluid. Asikainen et al.[123] reaffirmed the reliability and precision of the Periotron 6000 and demonstrated its insensitivity to qualitate differences in fluids collected.

Thus it is apparent that GCF flow is a reliable and sensitive indicator of gingival health and that the Periotron 6000 offers an objective method of GCF flow rate determination.

Methods and Materials

Selection of Subjects

The sample population consisted of 109 males, including 49 who used no tobacco of any kind and ranged in age from 18 to 71 years, with a mean of 27.41 years. Sixty were cigarette smokers, who ranged in age from 18 to 40 years, with a mean of 25.38. All subjects were recruited from military, dependent and retired personnel reporting for dental examinations or treatment at the Chanute Air Force Base Dental Services, Chanute Air Force Base, Illinois.

Each prospective subject was first screened by a member of the Chanute Air Force Base dental staff with the following criteria in mind: males 18 years old or older, nonsmokers or cigarette-only smokers, and with no medical or dental condition that might directly or indirectly affect the gingival tissues. Disqualifying systemic conditions included, but were not limited to, coronary heart disease and leukemia. Oral conditions included pathologic conditions, other than caries or periodontal disease, and local factors such as xerostomia, fixed partial dentures, orthodontic appliances, restorations with defective margins, or any other iatrogenic conditions that might produce gingival inflammation in the examination site (maxillary cuspid to cuspid) unrelated to the subject's smoking habit. Each subject's maxillary right and left central incisors, lateral incisors, and cuspids had to be minimally restored, especially in the gingival area. Prospective

subjects who indicated frequent use of any form of smokeless tobacco were excluded.

Prospective subjects meeting the above criteria were approached by the principal investigator and invited to participate. If the response was in the affirmative, each subject was asked to read and sign a consent form, complete a health history in order to verify his medical condition, complete a smoking questionnaire, and take the Fagerstrom Tolerance Questionnaire.[124] This questionnaire was developed to measure a smoker's degree of physical/psychological dependence on nicotine. It consists of eight questions designed to quantitate addiction: time from awakening to first cigarette; difficulty in abstaining; importance of the first cigarette of the day; number of cigarettes smoked per day; smoking rates in the morning; smoking during illness; brand smoked (nicotine content); and inhalation tendencies. Each question is scored, with higher scores given for responses indicating addiction, and the individual scores are added together to give a composite. The range of possible composite scores is 0-11, with 0 indicating minimal and 11 indicating maximum dependence. The questionnaire is commonly used in smoking cessation programs to identify those smokers who might benefit from a nicotine-containing chewing gum.[125,126]

If the subject had not smoked, eaten, or brushed and/or flossed in the examination area in the past hour, or the same area had not been disturbed by the aforementioned examination, the subject entered into the clinical phase of the study. Otherwise the subject was given an appointment at a later date and instructed not to smoke, eat, brush or floss for one hour prior to the appointment time.

Clinical Phase

This phase consisted of four examinations, in this order: Periotron recordings, determining the carbon monoxide concentration of expired air, recording a Plaque Index (PlI)[25], and recording a Papillary-Marginal Gingivitis Index (PMGI).[127]

Periotron Recordings

At least once daily, and as often as required, a Periotron 6000 (Harco Medical Electronic Devices, Inc., Irvine, CA) was calibrated according to manufacturer's instructions.

For each subject the maxillary six anterior teeth were isolated with two 1-1/2 inch sterile cotton rolls placed on the labial aspect of the teeth to be examined, just under the upper lip. The teeth and gingiva were dried with a gentle stream of compressed air from an air/water syringe, the excess fluid being captured by a cotton gauze gently placed on the lingual aspect of the teeth. Using the criteria of Silness and Loe,[25] plaque was scored on the facial aspect of the six maxillary anterior teeth only and then removed with an explorer, taking care not to disturb the gingival tissues. This was done so as not to compromise the overall PlI to be accomplished later.

Following the manufacturer's written instructions, the 5-second Tone Generator on the Periotron 6000 was activated. At the next tone a

Periopaper strip was inserted into the gingival crevice of tooth #6 until a slight resistance was felt. The strip was left in place five seconds (or until the next tone) and then removed and inserted between the Sensors of the Periotron 6000 until the alignment mark on the strip intersected the lower edge of the lower Sensor. The Sensor was then closed, thus activating a 16-second measuring cycle as evidenced by an illuminated Mode I lamp. When the Mode II lamp glowed, indicating the end of the measuring cycle, the number appearing on the digital readout screen was recorded on a specially designed form. The purpose of this initial strip placement was to clear the sulcus of excess fluid that might have accumulated during the isolation procedure.

A new strip was then placed in the same location and the procedure was repeated in order to measure the rate of gingival flow. The second number appearing on the digital readout screen was recorded. The technique was then repeated for teeth #7 through #11.

Carbon Monoxide (CO) Measurement

A MiniCO Model 1000 Carbon Monoxide Breath Instrument (Catalyst Research Corporation, Owings Mills, MD) was used to determine the concentration of carbon monoxide in expired alveolar air. At the beginning of each day and according to manufacturer's directions, the meter was calibrated by means of a calibration cylinder having 60 parts per million (ppm) CO.

Each subject, whether nonsmoker or smoker, was instructed to take a deep breath, hold it for 15 seconds, discard the first one-quarter of the expired breath by puffing gently, and then completely and forcefully exhale the remaining air into the analyzer's balloon through a plastic mouthpiece.

The digital readout, in ppm, subtracting the background or ambient CO, was recorded on the same form used for Periotron readings. The subject was then directed to another examiner for the next part of the clinical phase.

Plaque and Gingivitis Scoring

A board-eligible periodontist assigned to the Chanute Air Force Base Dental Services performed both the PII and the PMGI, and his observations were entered by a recorder on forms provided by the principal investigator. The periodontist had been previously calibrated in both indices at the Oral Health Research Institute, Indiana University School of Dentistry, Indianapolis, IN.

The Plaque Index of Silness and Löe[25] evaluates the presence or absence of plaque at the gingival margin of each tooth in the mouth and uses a scoring system of 0 to 3. Criteria are as follows: 0 = no plaque in the gingival area; 1 = a film of plaque adheres to the free gingival margin and adjacent area but can only be recognized by running an explorer along the tooth surface; 2 = moderate accumulation of soft deposits within the gingival sulcus, on the gingival margin and/or on

the adjacent tooth surface that can be seen with the naked eye; 3 = abundance of soft matter in these areas or a heavy accumulation in the interdental area. This index does not use a disclosing agent.

The PMGI involves the visual assessment of each papilla and gingival margin of all teeth, both labial and lingual, for the presence and severity of inflammation. Criteria are as follows: 0 = absence of inflammation; 1 = mild inflammation with slight changes in color and slight edema with no bleeding if probed; 2 = moderate inflammation with redness, edema, glazing, and bleeding if probed; 3 = severe inflammation with marked redness and edema and spontaneous bleeding.

Subject Dismissal

After all examinations were completed, the subject was, if he was a smoker, offered information on the smoking habit and quit-smoking programs in the area. At this point all subjects were excused, their participation having ended.

Results

As a preliminary step to a statistical analysis of the data, mean values for Periotron readings, plaque scores, and gingivitis scores were calculated for each subject. GCF flow rates for each tooth were added and the total was divided by the number of teeth examined. Likewise, mean PlI and PMGI scores were determined for both the entire mouth and, in the case of gingivitis, for the maxillary six anterior teeth. Teeth #6-11 were the same ones involved in GCF flow measurements.

All data were then evaluated with B-GRAPH (Batteries Included, Irvine, CA), a professional graphics-charting and statistical analysis program for the Atari 800 Personal Computer System (Atari, Inc., Sunnyvale, CA). Differences in GCF flow rates, as measured by the Periotron 6000, carbon monoxide concentration of expired air, PlI, and PMGI between smokers and nonsmokers were evaluated with two-tailed Student's t-distributions. In addition, selected pairs of factors were subjected to the Pearson correlation coefficient test to determine their relationship.

GCF Flow Rate

Table I summarizes the results of GCF flow rate measurements. Group means show that Periotron readings were numerically higher in smokers.

However, the magnitude of this difference was not statistically significant ($p < 0.60$). There were wide variations in flow rates in both groups, as evidenced by large standard deviations.

Carbon Monoxide

The carbon monoxide data are presented in Table II. As expected there was a higher concentration of carbon monoxide in the expired air of smokers than in nonsmokers, the difference being statistically significant ($t = 10.536$; $0.001 < p$).

Subjects with CO levels of less than 8 ppm are generally considered to be nonsmokers whereas those with values of 8 ppm or more are considered to be smokers. In this study 100% of the nonsmokers had CO values of 5 ppm or less. Ten smokers (16%) had CO levels of 7 ppm or less; 80% of these subjects smoked one-half pack of cigarettes per day or less, and 50% consumed less than 6 cigarettes daily.

Plaque and Gingivitis

Tables III and IV similarly summarize the results of dental plaque and gingivitis examinations, respectively. Both PII and PMGI scores were considered quite low in both smokers and nonsmokers. Nevertheless there was a statistically significant difference in both indices between the two groups.

From the group's mean PlI scores, a t-value of 5.604 was determined. This represented a highly significant difference ($0.001 < p$).

Similar results were found regarding PMGI. The difference in PMGI scores between the two groups yielded a t-value of 5.669, again a highly significant difference ($0.001 < p$).

Correlation of Selected Factors

Simple correlation (Pearson) analyses were conducted between selected pairs of quantitative data. Table V presents correlation coefficients between GCF flow rate and PMGI (teeth #6-11) in smokers and nonsmokers. Table VI presents similar data for GCF flow and PlI. An analysis of the relationship between PlI and PMGI (both full mouth) in smokers and nonsmokers is summarized in Table VII. Both GCF flow rate and plaque scores showed positive relationships with gingivitis, although plaque scores were more strongly correlated. GCF flow was weakly correlated with plaque scores.

A summary of the information obtained from the smoker's questionnaire and the Fagerstrom Tolerance Questionnaire is presented in Table VIII. The data include mean values and standard deviations for the number of cigarettes smoked per day, the number of years smoked, pack years, and Fagerstrom scores. Pack years are determined by multiplying the number of packs of cigarettes consumed daily by the number of years the individual has smoked.

Table IX presents the results of a series of correlation tests performed with data from the smoking group only. The following pairs of

factors were studied: GCF flow rate versus CO; GCF flow rate versus daily cigarette consumption; CO versus daily cigarette consumption; CO versus the number of years smoked; CO versus the Fagerstrom Tolerance Questionnaire score; CO versus PlI; CC versus PMGI; Fagerstrom Tolerance Questionnaire score versus daily cigarette consumption; Fagerstrom Tolerance Questionnaire score versus the number of years smoked; pack years versus the Fagerstrom Tolerance Questionnaire score; and pack years versus CO.

There was almost no correlation between GCF flow rate and either CO or daily cigarette consumption. There was a weak positive correlation between the Fagerstrom Tolerance Questionnaire score and the number of years smoked.

Somewhat stronger, but still only moderately positive correlations were found between CO and both Fagerstrom Tolerance Questionnaire scores and the number of years smoked, and pack years and Fagerstrom Tolerance Questionnaire scores.

Even stronger positive correlations were found between pack years and CO and CO and daily consumption of cigarettes. The strongest positive correlation was found between Fagerstrom Tolerance Questionnaire scores and the number of cigarettes smoked per day.

Slightly negative correlations were found between carbon monoxide and both plaque and gingivitis.

Tables

TABLE I

GINGIVAL CREVICULAR FLUID FLOW RATE
IN SMOKERS AND NONSMOKERS
AS MEASURED BY THE
PERIOTRON 6000

	N	MEAN	S.D.	T-VALUE	SIG
SMOKERS	60	8.691	5.644		
NONSMOKERS	49	8.110	5.100	0.553	NO

TABLE II

CARBON MONOXIDE CONCENTRATION
IN EXPIRED ALVEOLAR AIR IN
SMOKERS AND NONSMOKERS
(PPM)

	N	MEAN	S.D.	t-VALUE	SIG
SMOKERS	60	22.983	13.388		
NONSMOKERS	49	2.612	0.837	10.536	YES

TABLE III

PLAQUE INDEX (PLI) IN
SMOKERS AND NONSMOKERS

	N	MEAN	S.D.	t-VALUE	SIG
SMOKERS	60	0.652	0.339		
NONSMOKERS	49	0.319	0.261	5.604	YES

TABLE IV

PAPILLARY-MARGINAL GINGIVITIS
INDEX (PMGI) IN
SMOKERS AND NONSMOKERS

	N	MEAN	S.D.	T-VALUE	SIG
SMOKERS	60	0.751	0.281		
NONSMOKERS	49	0.407	0.347	5.669	YES

TABLE V

CORRELATION BETWEEN GCF FLOW RATE AND PMGI (TEETH #6-11) IN SMOKERS AND NONSMOKERS

	PEARSON'S CORRELATION COEFFICIENT
SMOKERS	0.507
NONSMOKERS	0.440

TABLE VI

CORRELATION BETWEEN GCF FLOW RATE AND PLI (TEETH #6-11) IN SMOKERS AND NONSMOKERS

	PEARSON'S CORRELATION COEFFICIENT
SMOKERS	0.383
NONSMOKERS	0.375

TABLE VII

CORRELATION BETWEEN PLI

AND PMGI IN SMOKERS

AND NONSMOKERS

	PEARSON'S CORRELATION COEFFICIENT
SMOKERS	0.679
NONSMOKERS	0.665

TABLE VIII

SUMMARY OF RESPONSES FROM SMOKER'S
AND FAGERSTROM TOLERANCE QUESTIONNAIRES

	MEAN	S.D.
#CIGARETTES/DAY	20.350	9.816
#YEARS SMOKED	8.341	5.761
PACK YEARS	9.333	7.839
FAGERSTROM SCORE	5.367	2.107

TABLE IX

CORRELATION BETWEEN VARIOUS

PARAMETERS IN SMOKERS

	PEARSON'S CORRELATION COEFFICIENT
GCF vs CO	0.026
GCF vs #CIGS/DAY	0.073
CO vs #CIGS/DAY	0.585
CO vs YEARS SMOKED	0.431
CO vs FAGERSTROM	0.481
CO vs PLI	-0.254
CO vs PMGI	-0.222
FAGERSTROM VS #CIGS/DAY	0.613
FAGERSTROM vs YEARS SMOKED	0.227
PACK YEARS vs FAGERSTROM	0.403
PACK YEARS vs CO	0.589

Discussion

The results of this study seem to suggest that cigarette smoking has little or no chronic effect on the production of gingival crevicular fluid. Mean flow rates for the smoking and nonsmoking groups were nearly identical, as were their standard deviations. There is only one other published report on the effects of smoking on GCF flow in man with which these results may be compared. Hedin et al.,[128] incidental to their study of cyclic nucleotide content in gingival tissue, found that gingival fluid flow was decreased in smokers. In a study analagous to the present one, Mendel and associates[129] used the Periotron 6000 to measure GCF flow response to smokeless tobacco products in Sprague-Dawley rats. They found a 2 to 3-fold increase in flow after subjecting the lower lip pouch to a 2-hour, twice daily exposure to chewing tobacco. It is speculated that the increase in flow produced by smokeless tobacco, which is placed in direct contact with the oral soft tissue, is due primarily to local effects. On the other hand, chronic cigarette smoking presumably affects the gingival tissues by a combination of local (heat, irritation) and systemic routes.

The finding of a significantly higher level of gingivitis in smokers in the present study, which is in agreement with a number of previously published reports[15,18,26,38,39] and is in contrast to others,[3,3,11,14,16,29,43] seems to be inconsistent with the aforementioned observations regarding GCF flow. The flow of fluid through the crevicular epithelium has been consistently associated with the severity of inflammation. As discussed earlier, several

investigators have suggested that GCF flow rate is a more sensitive indicator of gingival health than clinical indices based on subjective scoring, and that it permits earlier detection of changes in periodontal status.

These claims notwithstanding, the present study presents an apparent contradiction. If gingivitis is truly more prevalent in smokers, that fact would presumably be reflected in an increase in GCF flow as compared to nonsmokers, which was not the case here.

Perhaps the dichotomy can be explained by the pharmacologic action of nicotine, one of tobacco smoke's main constituents, on the circulatory system. In a recent study of a group of patients with peripheral vascular disease (P.V.D.), Laing et al.[130] found that over 90% of the study sample were smokers. Nicotine releases noradrenalin locally,[131] which increases the total peripheral resistance and reduces blood flow in peripheral vessels.[132,133] Lusby et al.[134] showed that smoking exacerbated the digital vasoconstriction normally seen in P.V.D.

Nicotine's action on peripheral vessels conceivably could manifest itself in the gingival circulation. The outward effects of the inflammatory response, e.g. bleeding and fluid flow, could be moderated by a narrowing of small vessels and a reduced rate of blood flow. Preber and Bergstrom[49,59] found that bleeding tendencies were markedly lower in smokers. They also postulated that their observations might be explained by the action of nicotine, citing the work of Shuler[52] and Clarke et al.[135] to support their hypothesis.

Bleeding tendencies were not evaluated in the present study. The PMGI[127] is a visual assessment only. Although the criteria contain

statements about bleeding, the scoring is based on clinical judgment as to whether the tissue, based on its color and contour, might bleed if probed. The examiner in this study did no probing. Regardless, the same forces may be operative regarding GCF flow. That is, the vasoconstriction and reduced blood flow suggested by Preber and Bergstrom[49,59] to explain reduced bleeding tendencies in smokers may also explain the nearly identical GCF flow rates seen in smokers and nonsmokers in the present study.

Thus, even though signs of inflammation were present (redness, edema, glazing, etc.) to a greater extent in the smoking group, the pharmaco-dynamic evidence of that inflammation (GCF flow) seemed to be suppressed.

Conclusions about plaque accumulation in smokers vary. Some studies[16-19,26,28] have found that smokers have more plaque; others[14,15,20,28,30,32] have found just the opposite. The results of the present study are consistent with the former group. Plaque scores were not only numerically greater, but the difference was also highly significant. Since no attempt was made to evaluate the frequency or efficiency of toothbrushing in either group, this remains an unknown etiologic factor in the present study. However, when Modeer et al.[19] and Macgregor[28] corrected for this variable, the differences they found remained significant.

That smokers have increased concentrations of carbon monoxide in expired air is a well-established fact. Vogt et al.[136] and Wald[137] have demonstrated that persons who smoke more than one pack of cigarettes daily had CO levels three times greater than nonsmokers. The actual

concentration depends on the cumulative effect of a number of variables such as the number of cigarettes consumed per day, the depth of inhalation, the number and volume of puffs taken per cigarette, the brand name of the cigarette (CO content varies between brands), etc.[138] While very low consumption can be reflected in CO levels below 8 ppm, as seen in the present study, this level is considered to be a reliable barometer to separate smokers from nonsmokers. While occasional smokers might have levels below 8 ppm, it is unlikely that nonsmokers would have levels approaching or exceeding 8 ppm.

The results of this study support others that have found significant differences in CO levels in expired air between smokers and nonsmokers. Carbon monoxide is also considered to play a role in the cardiovascular effects of smoking. Because of its high affinity for hemoglobin, carbon monoxide is responsible for the formation of carboxyhemoglobin (COHb). COHb reduces the oxygen-carrying potential of circulating blood and decreases the amount of oxygen released to tissues.[139] The concentration of CO in expired air has been repeatedly shown to be directly related to serum COHb percentages,[140-143] and thus it is a reliable measure for both chemicals.

In this study, carbon monoxide was negatively correlated with both plaque and gingivitis. This raises the question of whether carbon monoxide might have similar suppressive effects on the growth of selected plaque bacteria and peripheral circulation. Because the correlations were weak ones, this remains a matter of conjecture and a subject for further research.

The results of this study support the use of both CO measurements and the Fagerstrom Tolerance Questionnaire to verify tobacco consumption and to determine nicotine dependence. Assuming the responses to questions on the smoker's questionnaire to be accurate, both CO levels and Fagerstrom scores were highly correlated with daily cigarette consumption and with each other. CO was equally correlated with pack years. The findings indicate that both techniques are useful in smoking-cessation programs for their verification and motivation potential.

Summary and Conclusions

In this study there were no differences in gingival crevicular fluid flow rates in smokers and nonsmokers. Chronic tobacco smoking seemingly had little or no effect,although the possibility exists that nicotine's vasoconstrictive effects suppressed fluid flow.

There were highly significant differences in carbon monoxide concentrations in expired air. As expected, smokers had markedly higher levels.

Smokers had significant higher plaque (PlI) and gingivitis (PMGI) scores than nonsmokers.

GCF flow rates, as measured by the Periotron 6000, showed a positive correlation with PMGI scores, but it was not as strong as the correlation between PMGI and PlI scores. Flow rates showed a somewhat weaker positive correlation with PlI. GCF flow did not correlate with either carbon monoxide or daily cigarette consumption.

Carbon monoxide readings in smokers showed positive correlations with daily cigarette consumption, the number of years smoked, pack years, and Fagerstrom scores, but negative correlations with both PlI and PMGI.

Fagerstrom scores showed, in order of decreasing strength, a positive correlation with the number of cigarettes smoked per day, pack years, and years smoked.

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Appendix

APPENDIX I

Subject #_____

CONSENT FORM

Dear Panelist,

In partial fulfillment of the requirements for a Master's Degree in Preventive Dentistry from Indiana University School of Dentistry, I am conducting a study to determine if cigarette smoking causes or contributes to the causes of early gum disease. Many studies have been done in this area, but the results have been inconclusive. My hopes are that the unique design of the present study will help to clarify the situation and to determine if the occurrence of gum disease can be linked to cigarette smoking.

At the beginning of the study you will be asked (1) to read and sign this consent form indicating that you understand what will happen in the study and that you volunteer to participate; (2) complete a health history form; and (3) complete two smoker's questionnaires. You will then be given a dental examination, without x-rays. If you do not have a medical or dental condition that would disqualify you, and if you are either a non-user of tobacco in any form or a smoker of cigarettes only, you will be invited to return to the Chanute AFB dental clinic for a follow-up appointment, the time and date of which to be determined according to your schedule. Disqualifying conditions include, but are not limited to, severe heart disease, leukemia, any oral disease other than tooth decay or gum disease, the presence of crowns (caps) or bridges (false teeth) in certain areas of your mouth, or the absence of teeth in these same areas.

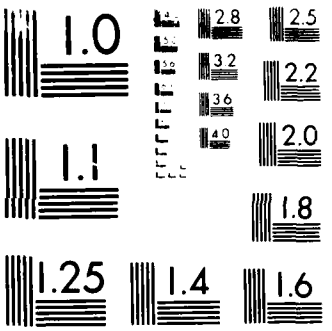
You will be asked to not brush your teeth, floss your teeth, eat, drink, or smoke for at least one hour prior to the second appointment. At the second appointment we will be measuring the flow of tissue fluid from around selected teeth. The areas selected will be isolated with cotton rolls. A small piece of filter paper will be placed in the space between each tooth selected and the adjacent gum tissue. Each piece of filter paper will be left in place for five seconds, after which it will be placed in an electronic device that measures the amount of fluid collected. This test will be performed twice on a sample of six teeth for a total of twelve measurements. Afterwards the carbon monoxide content of your breath will be measured by asking you to exhale into a special balloon. Another dentist will then make a visual assessment of your gum's health status and the amount of plaque (debris) on your teeth. This will complete your participation in the study. Time required for the second appointment is estimated to be between 20 and 45 minutes.

The risks to you in this study are minimal and are no greater than a routine dental examination. Feel free to ask me any question or questions about your possible participation.

If you wish to participate, please sign this form, complete the health history and the smoker's questionnaires. You will then be given a day and time for the second appointment. It is stressed that your participation is strictly voluntary and that you may withdraw at any time without prejudice. While the overall results of this study may be published in a scientific journal at some later date, your identity will remain in strict confidence. It is estimated that approximately 225 individuals will participate in this study. You will incur no expenses as a result of your participation, nor will you receive any payment. The investigators assume no responsibility for your dental condition.

AD-A171 965 THE EFFECT OF CIGARETTE SMOKING ON GINGIVAL CREVICULAR FLUID FLOW(U) AIR FORCE INST OF TECH WRIGHT-PATTERSON AFB OH L P CRIGGER 1986 AFIT/CI/NR-86-164T 2/2

UNCLASSIFIED F/G 6/5 NL


1.0
1.1
1.25
2.8
3.2
3.6
4.0
2.5
2.2
2.0
1.8
1.4
1.6

If you wish, at the end of the second appointment, we can provide you with information about quit-smoking programs. If you have any questions or wish further information, please call me or leave a message for me at the Chanute dental clinic (495-3902).

Sincerely,

LtCol Laurence P. Crigger, D.D.S.

Graduate Student, Indiana University

I have read and understand the above information.

_____ _____

Panelist signature date

_____

Witnessed by

APPENDIX II

Subject #_____

HEALTH HISTORY QUESTIONNAIRE

Dear Panelist:

As part of this study, we would like for you to complete this health history form. A completed form is required for every person who participates in the study. All correspondance is held in complete confidence and is retained for only as long as you are an active participant. **PLEASE PRINT**

Name _____Date of Birth_____Age_____

Last First M.I.

Address_____

City (Base)_____ Zip_____

Phone (Home)_____ (Work)_____

Describe your general health: Good___ Fair___ Poor ___

==========

1. Have you ever had one or more of the following serious illnesses or conditions that required hospitalization or a physician's care? Yes___ No___ (Circle those that apply)

Severe heart disease Heart attack Leukemia

High blood pressure *Hyperthyroidism* Stomach ulcer

2. Do you presently have a serious health problem? Yes___ No___ If yes, please describe:

_____

3. Have you taken drugs/medications during the past year or are you taking any now? Yes___ No___ If yes, please list:

_____

4. Have you ever had any of the following? If yes, check the appropriate space and explain below.*

__ALLERGIES	__BREATHING PROBLEMS	__HEPATITIS	__RHEUMATIC FEVER
__ANEMIA	__DIABETES	__JAUNDICE	__TUBERCULOSIS
__ASTHMA	__EPILEPSY	__KIDNEY DISEASE	__TUMORS OR GROWTHS
__BLEEDING DISORDERS	__FAINTING SPELLS	__LIVER DISEASE	__VENEREAL DISEASE
	__HEART TROUBLE	__NERVOUSNESS	__AIDS OR HERPES

*Explanation _____ _____

_____

==========

I have read the description of the dental study and wish to participate in the program.

_____ _____

Panelist signature Date

_____

Witnessed by

Reviewer Disposition _____

APPENDIX III

Subject #_____

SMOKER'S QUESTIONNAIRE

Panelist Name_____

Dear Panelist:

Thank you for participating in this study. We believe that this study will add to the body of scientific knowledge regarding the relationship between smoking and oral diseases. We would like you to complete the following questionnaire.

1. What forms of tobacco do you smoke and approximately how much or how many do you smoke per day?

_____ cigarettes	_____ number per day
_____ cigars	_____ number per day
_____ pipe	_____ bowls per day
_____ other	_____ number per day

_____ I DO NOT USE TOBACCO PRODUCTS

2. Name the brand of cigarettes you currently smoke. Circle "F" if it is a filter cigarette and/or "M" if it is mentholated.

_____ F M

Brand Name

3. What size are your cigarettes?

__ regular __ king __100 mm __120 mm

4. Do you inhale?

__ never __ sometimes __ always

5. If you are a cigarette smoker, approximately how many years have you been smoking?

_____

6. If you are currently a smoker, when did you smoke your last cigarette?

_____ hours ago

7. Do you use any form of smokeless tobacco (e.g., snuff, chewing tobacco, plug tobacco) ___Yes ___No

If yes, please answer the following questions:

a. What forms of smokeless tobacco have you used and how much per day do you use?

___ snuff	___ number of dips/day
___ chewing tobacco	___ number of chews/day

___ plug tobacco ___ number of chews/day

b. What brands of smokeless tobacco do you use?

_____

c. How long have you been using smokeless tobacco products?

_____

8. If you now smoke, would you like information about quitting?

_____ _____

Panelist signature Date

_____

Witnessed by

APPENDIX IV

Subject #_____

THE FAGERSTROM TOLERANCE QUESTIONNAIRE

Panelist Name_____

Question	Score*
1. How soon after you wake up do you smoke your first cigarette? __Within 30 min __After 30 min	_____
2. Do you find it difficult to refrain from smoking in places where it is forbidden, e.g., in church, at the library, cinema, etc.? __Yes __No	_____
3. Which cigarette would you hate most to give up? __the first of the day __the last of the day __other	_____
4. How many cigarettes a day do you smoke? __less than 15 __15-25 __more than 25	_____
5. Do you smoke more frequently during the morning than during the rest of the day? __Yes __No	_____
6. Do you smoke if you are so ill that you are in bed most of the day? __Yes _No	_____
7. Do you inhale? __Yes __No	_____
8. What brand do you smoke? _____	_____
TOTAL	_____

_____

Witnessed by

* Score to be filled in by examiner.

Subject #_____

PERIOTRON RECORDINGS

UPPER FACIAL

	6	7	8	9	10	11
1ST						
2ND						
DIFFERENCE						

SUBJECT MEAN

CARBON MONOXIDE READING

Subject #_____

PLAQUE INDEX

UPPER FACIAL

RIGHT LEFT

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
D F M	D F M	D F M	D F M	D F M	D F M	D F M	D F M	M F D	M F D	M F D	M F D	M F D	M F D	M F D	M F D
D L M	D L M	D L M	D L M	D L M	D L M	D L M	D L M	M L D	M L D	M L D	M L D	M L D	M L D	M L D	M L D
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16

UPPER LINGUAL

LOWER FACIAL

RIGHT LEFT

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17
D F M	D F M	D F M	D F M	D F M	D F M	D F M	D F M	M F D	M F D	M F D	M F D	M F D	M F D	M F D	M F D
D L M	D L M	D L M	D L M	D L M	D L M	D L M	D L M	M L D	M L D	M L D	M L D	M L D	M L D	M L D	M L D
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17

LOWER LINGUAL

APPENDIX VI

Subject #_____

PMGI INDEX

UPPER FACIAL

RIGHT LEFT

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
D F M	D F M	D F M	D F M	D F M	D F M	D F M	D F M	M F D	M F D	M F D	M F D	M F D	M F D	M F D	M F D
D L M	D L M	D L M	D L M	D L M	D L M	D L M	D L M	M L D	M L D	M L D	M L D	M L D	M L D	M L D	M L D
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16

UPPER LINGUAL

LOWER FACIAL

RIGHT LEFT

32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17
D F M	D F M	D F M	D F M	D F M	D F M	D F M	D F M	M F D	M F D	M F D	M F D	M F D	M F D	M F D	M F D
D L M	D L M	D L M	D L M	D L M	D L M	D L M	D L M	M L D	M L D	M L D	M L D	M L D	M L D	M L D	M L D
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17

LOWER LINGUAL

Curriculum Vitae

Laurence Paul Crigger

July 11, 1949	Born in Huntington, West Virginia
September 1967 to June 1970	Marshall University (no degree) Huntington, West Virginia
March 25, 1970	Married to Sally Elaine Bethel
May 1974	D.D.S., West Virginia University Morgantown, West Virginia
August 1974 to February 1977	Base Dental Officer McGuire Air Force Base, New Jersey
February 1977 to July 1981	Base Dental Officer Chief, Restorative Dentistry Chief, Endodontics Eielson Air Force Base, Alaska
July 1981	Named Outstanding Dental Officer Alaskan Air Command, 1981
July 1981 to August 1984	Chief, Dental Consultation USAF Dental Investigation Service USAF School of Aerospace Medicine Brooks Air Force, Texas
July 1982	Fellow, Academy of General Dentistry
July 1982	USAF Commendation Medal
August 1984 to June 1986	M.S.D. Preventive Dentistry Indiana University School of Dentistry Indianapolis, Indiana
January 1985	USAF Meritorious Service Medal
July 1986	Assigned to Ramstein AB, Germany

Professional Organizations

American Dental Association
Academy of General Dentistry
International Association for Dental Research

Abstract

The Effect of Cigarette Smoking on Gingival Crevicular Fluid Flow

by

Laurence Paul Crigger

Indiana University School of Dentistry
Indianapolis, Indiana

Gingival crevicular fluid (GCF) flow rates were measured with a Periotron 6000 in 60 smokers and 49 nonsmokers. In addition, carbon monoxide (CO) concentration of expired air was measured, and plaque and gingivitis indices were recorded for all subjects. All subjects completed a medical history and a smoker's questionnaire. Smokers also completed the Fagerstrom Tolerance Questionnaire.

Differences in GCF flow between smokers and nonsmokers were not statistically different. Smokers had a higher concentration of CO in expired air, more plaque accumulation, and a higher gingivitis score than nonsmokers. The differences in all three parameters were highly significant.

GCF was positively correlated with gingivitis scores, but plaque scores showed a stronger correlation in both groups. GCF showed no

correlation with either carbon monoxide levels or the number of cigarettes smoked per day.

There were strong positive correlations between Fagerstrom scores and daily tobacco consumption, as well as between carbon monoxide levels and both daily consumption and lifetime consumption as measured by pack years. Still positive, but slightly weaker correlations were found between GCF flow and plaque accumulation, between carbon monoxide levels and Fagerstrom scores, between carbon monoxide levels and years smoked, and between Fagerstrom scores and both pack years and years smoked.

Negative correlations, albeit weak ones, were found between carbon monoxide and both plaque and gingivitis prevalence.

END

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